UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGIONS 5
77 WEST JACKSON BOULEVARD
CHICAGO, IL 60604-3590

September 1, 2006

Mr. Jerry C. Winslow
Principal Environmental Engineer
Xcel Energy
414 Nicollet Mall (Ren. Sq. 8)
Minneapolis, Minnesota 55401

REPLY TO THE ATTENTION OF:

SR-6J

RE: Comments on the Draft Baseline Ecological Risk Assessment
Ashland/NSP Lakefront Superfund Site

Dear Mr. Winslow:

The United States Environmental Protection Agency (EPA) has completed its review of the draft Baseline Ecological Risk Assessment submitted on behalf of Northern States Power Company/Xcel Energy by URS on May 30, 2006 for the Ashland/Northern States Power Lakefront Superfund Site. Our comments are provided below:

General Comments

1. It is our understanding that the BERA would incorporate data collected for the 1998 and 2001 ERAs performed by SEH. While SEH data was used to select COPCs and to propose sediment cleanup levels, it appears that this data was not incorporated into the food chain modeling. Fish tissue from SEH study should also be included.

2. We agree that site sediments should be addressed in the FS. However, the impacts from soil and sediments to higher level organisms has not been adequately characterized in the BERA, so conclusions on excluding these pathways is premature at this time.

3. The BERA does not appear to address the free product in the bay area. Therefore, it is assumed that removal of the free product will be addressed in the RAOs and FS.

4. The shallow groundwater discharge to the bay area does not appear to have been addressed.

5. We do not concur at this time that the relative significance of the lines of evidence presented in Section 4.3.2 (numbers 1 through 3) are appropriate for this site. Further characterization of site risks and the uncertainty associated with each line of evidence needs to be performed before relative significance is assigned to a line of evidence.

6. Wood chips were commonly used in the purification process at MGP sites. Therefore, without documentation and proof that the wood chips from the purification process were not disposed of in the ravine and in the lower bluff and harbor area, it cannot be concluded that the wood chips are present solely due to non-MGP processes (lumber operation).

7. When presenting total PAH concentrations and normalized organic carbon (NOC) PAH concentrations, the associated organic carbon content should also be presented.

8. The shallow soil exposure point concentrations (EPCs) used in the human health risk assessment (HHRA) for recreational exposure in Kreher Park differ from the EPCs used to evaluate soil exposure by the mouse and blackbird. Why do the datasets differ?

9. Ecological RAOs presented in the RI/FS (Appendix A) will need to be adjusted after the BERA is corrected.

10. The BERA repeatedly states that laboratory test conditions fail to adequately represent the conditions present at the Site (especially in terms of UV light). While this does produce some uncertainty in the risk analysis, it does not necessarily mean that lines of evidence based on laboratory testing should be given "very low weight" in the weight-of-evidence approach, especially since modeled exposures were used for several measurement endpoints.

11. The risk assessment itself is very long winded in its general overview of the fate and effects of various COPCs, but comparatively slim in its actual evaluation of the site data. As an example, an extensive sediment toxicity testing effort is simply summed up in a table of NOECs and LOECs. Pages of discussion of UV effects are dismissed with a simple "that's an uncertainty, not an effect." There is little attempt to integrate different information into a more thorough assessment. For example, the bioavailability information provided by the bioaccumulation testing is not considered as a tool to help interpret the results of the sediment toxicity tests. It is very surprising that nowhere in this document or the appendices is there a single graph showing the relationship between a toxicity parameter and sediment chemistry. We would have thought that would be the first thing to do, and much more robust than the (sometimes arbitrary) assignment of NOEC/LOEC values. Relying solely on NOEC/LOEC values from hypothesis testing rather than looking at exposure/response relationships seems like a step backwards in risk assessment methodology.

12. With regard to effects on the benthic community, the risk assessment concludes that there is evidence for risk, which we agree with. However, we do not agree with several aspects of the analysis that are used to estimate risk thresholds. If all that is needed from this document is a decision as to whether there is risk to benthos, then the details of the risk estimation methods may not need to be decided now. However, if the analyses in this risk assessment are to be the basis for deriving cleanup goals, then the analysis needs revision and possibly supplementation, depending on the level of resolution needed to design remedial actions.

13. The sediment toxicity analysis is overly simplistic and arrives at risk thresholds well above concentrations shown to be dramatically toxic to benthic organisms. The same data could be used to justify a risk threshold roughly 10 times lower than what the authors have proposed. See Figure 1.

14. The authors elected to eliminate UV-induced PAH toxicity from the effects analysis. There is little meaningful justification for this. It should be included as part of the analysis; the authors concerns about the applicability of this information can be addressed in the uncertainty discussion. At present, not only has UV-induced PAH toxicity been removed from the effects discussion, it is not even discussed as an uncertainty.

15. The accumulation data for *Lumbriculus* are not used to their full effectiveness, as a means to explore bioavailability issues that could underlie all of the benthic community assessment. Some of these data contradict assertions made in the body of the assessment.

16. There is a heavy emphasis on the benthic community study as being the "strongest" line of evidence and not providing clear evidence of in situ effects on the benthic community. While the conceptual rationale for this is reasonable, it assumes the study has the discriminatory power to detect differences. The degree of variability observed, both within and between sampling locations, brings this very much into question. If the benthic community study has low power, then it is prone to underestimating effects and is in fact a weak line of evidence rather than a strong one.

Sediment Toxicity Testing

17. The range of PAH concentrations in the authors studies did not, unfortunately, succeed in providing a good range of contamination near the effect threshold. There is a better than 20-fold gap in PAH concentration between SQT7 and SQT3 which bracket the purported threshold.

18. The authors use a result from the 10-d sediment dilution study to establish the NOEC for *Hyalella*. This seems inappropriate given that it is mixing 10-d and 28-d results, and other samples in the 10-d dilution study with PAH concentrations higher than SQT7 do not show effects, indicating that the sensitivity of the 28-d test is greater than the 10-d test, as might be expected. It also ignores an effect concentration of around 1500 ug/g OC found by SEH (2002). Because of the large gap in concentrations, we would assert that the threshold for effects is highly uncertain based on the available site-specific data. The text should be revised to reflect this uncertainty. Because sediment toxicity appears to be one of the main lines of evidence establishing the existence of risk, resolving this uncertainty may be a high priority for the FS.

19. The authors chose to ignore data from "wood" stations in their derivation of thresholds for sediment toxicity. The stated reason for this is the belief that the wood matrix would be a poor sorbent for PAHs and, as a result, the organic-carbon normalized effect concentrations would be inappropriately low. While this is conceptually reasonable, it is not at all supported by the data. Bioaccumulation testing with *Lumbriculus* indicates that the bioavailability of PAHs in "wood" sites is very comparable to that in "sand" sites (see Table 1, attached). Accordingly, there is no evidence that PAH bioavailability misrepresented by OC-normalized PAH concentrations at wood sites. This is also important because it moves the LOEC

down to circa 1500 ug/g OC with the inclusion of the Wood #1 site from the SEH (2002) study. This suggests three-fold greater risk than is indicated by the current analysis.

20. The authors proposed LOEC/NOEC values do not seem well founded when one looks at the totality of the site sediment toxicity testing done by either SEH or URS. This is shown in Figure 1. What one sees is a) there is evidence for toxicity at PAH concentrations considerably below the LOEC/NOEC proposed by the authors; b) there is a large range of PAH concentrations not represented by the data in hand; and c) there is nothing in the site data to suggest that the risk benchmark proposed by EPA using equilibrium partitioning is not applicable to the site. This EPA value is 5 to 10 times lower than the concentrations suggested by the authors (the range is created by assumptions regarding the role of unmeasured PAHs; lower bound of EPA estimate assumes that priority pollutant PAHs comprise about half of the total PAH mixture, based on other work at coal tar sites [see publications by Kreitinger et al.]).

21. Although it is not justified by the data, if the risk assessment discounts wood stations in evaluating risk, how would clean-up goals for areas with wood substrate be determined? Further, why would one include wood stations in the community analysis if one can't relate chemical concentration to expected risk?

22. How can SQT3 be a sand station and have 40% organic carbon? Was the composition of the organic carbon in this sample verified? This is the OC range observed in wood stations; it does not sound like sand. This may be particularly important since SQT3 seems to be on the borderline of toxicity, even though the apparent OC-normalized PAH concentration is not that high. If this station had an OC concentration more typical of a sand station, then this might make sense.

23. Table 5-3 proposes a "no effect" level of 7257 ug/g OC for *Hyalella*. This needs to be clarified. The authors own tests show that a sediment with 6090 ug/g OC caused between 83 and 94% mortality. In addition, SEH found a sediment with 1580 ug/g to cause 97% mortality. This is in no way a "no effect" concentration.

24. The failure of the authors to produce additional data on the toxicity of sediments to *Chironomus* leaves the assessment to rely only on previous data. In discussing these data (page 5-18) the authors imply some uncertainty about effects at 3900 ug/g OC. However, it's worth pointing out that there was also 100% mortality at 4800 ug/g OC – so if anything, it's surprising that there wasn't more mortality at 3900 ug/g OC, which is the opposite of questioning the existence of effects at 3900 ug/g OC.

25. There are several references to spurious effects in the sand reference stations (e.g., 5-17), implying that site sediments may be affected by regional background contamination in addition to site-related contamination. However, it's worth noting that there was also indications of poor performance in some laboratory control treatments (formulated sediments) and, more significantly, there was no indication of spurious toxicity in sandy site sediments with lower PAH contamination. From this, it is not at all clear whether the results found in sand reference sediments have import to the assessment of site-related risks.

26. On Page 6-4, the conclusions regarding risk levels for benthos are inappropriate. First, as shown in Figure 1, the interpretation of the *Hyalella azteca* data are such that the proposed NOECs would be higher than concentrations that caused near complete mortality. The suggestion that the *Chrionomus* test procedure is inadequate for assessing effects is concerning, as it has been evaluated through round robin testing and widely applied. I have no idea why the authors had difficulty conducting the test, but that does not mean that the species is irrelevant to the risk assessment. More importantly, this decision ignores data presented by SEH that clearly shows adverse effects at concentrations below the suggested NOEC values. Finally, the analyses by Ingersoll et al. on Great Lakes sediments used chronic *Hyalella* data but only 10-d exposures for midge, which may explain part of the trend they observed.

Photoactivated Toxicity

27. The authors have chosen to treat UV/PAH effects as an "uncertainty" rather than a component of the risk assessment. While we don't agree with this decision, the authors do not include this issue in the uncertainty analysis, particularly with the notation that this omission errs entirely on the side of underestimating effects.

28. One of the reasons given for discounting photo-activated PAH toxicity is that the UVB levels in the laboratory exposures were higher than those measured in the field. We are not sure from this comment whether the concern is that UVB levels were high enough to cause direct phototoxicity, or the excess UVB would contribute substantially to the photo-activation of PAHs. In either event, it seems a little odd for the authors to dismiss the studies as inappropriate when they were themselves responsible for designing and executing it. While we would accept that excess UVB would create an uncertainty in applying the study results, it is not at all clear that it is a legitimate basis for disregarding the entire mechanism.

    It was also a little surprising that the lab light source would have that high a UVB intensity after filtering through glass, although UVB removal by glass is dependent on the thickness of that glass (not specified) and to some degree on its composition. In our laboratory exposure system, which uses the same UVA-340 bulb, we made measurements using the same model International Light 1700 meter used in the Ashland study. Inserting a piece of 1/8" frosted window glass reduced UVB to only 6.6% of its original intensity, while UVA was only reduced to about 30%. Put differently, the ratio of UVA to UVB without glass filtration was 3500:292 or about 8:1 (which is similar, but slightly higher than the roughly 10:1 ratio in sunlight). After passing a 1/8" sheet of window glass, this ratio 1066:19.3 or about 55:1, which is much higher than the 12:1 ratio measured during the laboratory UV assays. While the thickness of the glass used as covers is not described, it is very likely that using thicker glass filters could bring the ratio closer into line with nominal.

29. While it ends up having little impact on the final UV levels used in the UV studies, the analysis used to derive the UV exposures contains errors. Most significantly, the authors used an average of readings taken at 1000 and 1400 hours to create an estimate for 1200 hours. Because solar noon is at approximately 1300 hours at

Ashland under Daylight Savings Time, and because solar irradiance is for all practical purposes symmetrical around solar noon, the 1400 hour readings would have been the appropriate estimate of the 1200 hour readings rather than the mean of 1000 and 1400. The estimated clear sky irradiance curve was re-modeled after correcting this error, and obtained Figure 2. While this curve lies above the curve used by the authors, a more detailed algorithm was used to estimate total daily UV dose, by using the same basic approach as the authors, but using 0.1 hour time steps. This more detailed averaging altered the total UV exposure estimate by the nearly exact opposite amount as recalculating the daily irradiance curve. The net effect is that recalculated values were very close to the target values used originally derived. It is brought up here only for the record in case similar calculations are used later in the RI/FS process.

30. The depth for which UV exposure was estimated is listed in the LSRI report as being for SQT2, the deeper of two stations evaluated, but we believe it was actually for 232 cm depth, which was that for SQT1, the shallower station. As explained at length in the background material, incident UV is a function of depth, and the potential for photoactivated toxicity varies with depth accordingly. This can be incorporated into the analysis using a reciprocity assumption, discussed in the background material and well supported by experimental work. This relationship says in essence, that if you double the UV exposure, the PAH body burden associated with toxicity will be halved. Alternatively, if you halve the UV exposure, the PAH body burden required for equivalent effect will be doubled.

    Potency of the Site PAH mixture can be estimated by comparing the responses in the dilution studies of SQT1, which was tested under lab light, under UV light, and under UV light with leaf plugs added as a potential source of shading. The LC50 and LC20 concentrations can be estimated independently for each, as shown Table 2. See Attachment 1 for further details on this analysis.

    As narcosis and photo-activated toxicity occur by different mechanisms, the sediment toxicity under lab light can be reasonably assumed to be independent from the photo-activated toxicity. As such, one can estimate the effect of photo-activated toxicity alone by removing the binomial probability of mortality under lab light from that under UV light (with or without shading). These estimated values are also shown in Table 2. This analysis indicates that the presence of leaf plugs reduced the effective UV exposure by about 40%, but UV exposure was not eliminated.

    From these corrected EC20 values, one can estimate the sediment PAH concentration that would be associated with a 20 percent effect at depths other than the one simulated by the lab assays (SQT1). These values are shown in Table 3. While little additional toxicity from photo-activation would be expected at stations deeper than SQT1, for areas of the site shallower than SQT1, risk thresholds for PAH contamination could be expected to decrease dramatically. Development of remedial goals for areas substantially shallower than 232 cm may need to consider more rigorously the influence of photoactivated toxicity in establishing risk to benthos.

31. Somewhere in the document or its appendices it was suggested that there was inconsistency in response between the UV studies reported by SEH and those conducted by the authors. The data was reanalyzed and it was found not to be true. The PAH concentration data for the SEH data are confused by what appear to be spurious OC measurements (OC contents are not monotonic across the sediment dilution series). Problems with sediment OC measurements are not uncommon, particularly when the analytical instrument uses small sample sizes and the sediments contain large organic particles. However, if one simply expresses exposure as percent sediment, and then models the LC50 measured in the presence and absence of UV light, one gets a ratio of 3.4 for the sand series and 3.7 for the wood series. After correcting for the slightly higher UV in the SEH studies, one would expect a ratio of 2.6 based on the URS dilution studies. We would argue this is a pretty good agreement, rather than inconsistency.

32. The LSRI report also states that the modeled UV assumption was that the sky was 75% clear. While the 75% correction is accurate, the rationale is slightly different. The assumption is that the effect of cloud cover and other weather would, over time; result in an average incident UV that is approximately 75% of the clear sky value. As such, this estimate is an average of expected exposure, not a worst case. The worst case (10 consecutive sunny days) would be expected to be roughly 1/3 more potent than the condition tested.

33. Section 5.1.2.1 lists (bottom of page 5-10) UVB as the most damaging UV radiation. This is true for some toxic mechanisms, but not all. This distinction is further modified by the specific environment, such as for the example of water column attenuation discussed here. The mix of mechanisms discussed with respect to UV create problems with generalizations like this.

34. Page 5-15 – "In field samples, it may be difficult to separate the effects of UV light and PAHs from those of the multiple environmental contaminants inevitably present." Why any more so than any other endpoint? In the studies conducted, the relationship of toxicity enhancement by UV and sediment PAHs seems pretty clear.

35. Page 5-15 – "Therefore, the ability to create photosensitivity under laboratory conditions does not necessarily mean that toxicity will occur under natural conditions." This text seems to be downplaying the relevance of laboratory exposures to estimate effects in the field, but the mechanisms being discussed immediately previous to the statement (partitioning, burrowing into sediment) are operative in laboratory studies of the type conducted in this study, and are therefore not relevant to the issues at hand.

36. Bottom of page 5-15 – this discussion relies heavily on the McDonald and Chapman paper, the substance of which is far from a consensus viewpoint. More specifically, this discussion repeats arguments used by McDonald and Chapman which relied on quotes used without their original context in a way convenient to the arguments McDonald and Chapman were anxious to advance. For example, the Swartz study involved primarily deepwater sites, where one would not necessarily expect photoactivation because of the combined effects of water column attenuation and the

burrowing habits of these particular amphipods. McDonald and Chapman present this as though photoactivated toxicity was expected but not observed. The quote from the Diamond paper does not actually appear in the published manuscript (Diamond et al. 2003). Although it was present in an earlier draft provided as a courtesy to Dr. Chapman, the quote as presented in their paper is also taken out of context; the adjoining text in the manuscript provided them went on to discuss the fact that exposure used in the experiments was not the same as that received in the field, but that other organisms with comparable sensitivity but higher UV exposure may be at risk. For the amphipods at that particular site, lower PAH exposure and high water column attenuation would be expected to protect these organisms; these factors have been accounted for in the study design used for the Ashland site, so the statements by Diamond et al. are not relevant to the situation at hand.

37. Section 6.2.1.4 and 6.2.2.3 – Dismissal of a line of evidence should require something more than vague references to beaker size effects (not discussed anywhere else). This is particularly true when sediment testing included the provision of refugia (leaves). While the UVB may be an uncertainty, its seriousness is not defined and, further; it errs to the conservative side as the rest of the document touts as the way uncertainties were handled. This is not an appropriate justification for discarding this line of evidence.

38. Table 6-4 – the discussion of uncertainties does not include the potential underestimation of risk arising from discounting the potential for photoactivated toxicity.

***Lumbriculus* bioaccumulation study**

39. The document asserts that wood chips present in some sites exaggerate bioavailability; the data from the accumulation study does not bear this out, as BSAFs at wood sites are comparable to those at sand sites.

40. The risk assessment asserts that using site-specific BSAFs is a "conservative" assumption. It would seem that these site specific BSAFs are in fact the best estimate of the true value, not a conservative assumption.

41. There is mention that soot carbon data were collected, but I did not see that in the documents. These data should be compared to the bioaccumulation data to see how they relate to observed bioavailability. Only one site, SQT8, shows accumulation consistent with of having bioavailability being affected by black carbon (total PAH BSAF = 0.15); all others have BSAF>1.

42. The PAH coverage in this study included only a couple alkylated PAHs. Other data (see papers by Kreitinger and co-workers) suggest that in other coal gasification waste sites, alkylated PAHs make up roughly half of the total PAH mass. To account for this, the NEBR should be divided by 2, or some other appropriate correction factor.

43. EPA's sediment ESB for PAH mixtures used only PAH toxicity data in an analysis

similar to DiToro et al and arrived at an NEBR specifically for PAHs that is approximately 40% lower than DiToro et al. This is not acknowledged in the document.

44. Page 6-5 – The NEBR is exceeded beyond SQT1 and SQT7, particularly if you reduce the NEBR to account for unmeasured chemicals and/or use the EPA ESB NEBR instead of DiToro et al.

45. Page 6-23 – Text regarding the bioaccumulation study suggests that the site specific bioaccumulation studies are somehow questionable because BSAFs greater than 1 were found. The DiToro estimates are not bounds on theoretical possibility, they are estimates based on specific assumptions regarding partition coefficients. If the partition coefficients at this site are reduced, such as by the presence of wood as suggested by the authors elsewhere in the document, then these BSAFs are not only within theoretical possibility, they are the theoretical expectation.

Benthic community study

46. The text emphasizes that the benthic community study is the most important line of evidence because it incorporates real world exposures and the actual invertebrate community exposed. While these conceptual arguments are true, the ability of a benthic community study to represent these qualities lies in the ability of the study to detect differences, if they exist. If a study encounters a large degree of variability such that discriminatory power is greatly decreased, then the strength of the benthic community study as a line of evidence is decreased commensurately. It appears that there was tremendous variability encountered (which is not unusual), even in the PAH exposures. Plots included in the Appendix 2 indicate that the range of PAH concentration measured at SQT 1 overlapped the range of most or all site locations (incidentally, log scales would be a big help in displaying data with large ranges in values). With that kind of heterogeneity within a sampling location, is it really reasonable to expect much discriminatory power in associating exposure with community condition? Further, there was considerable scatter among reference stations, which would further cloud detection of community differences. There is neither discussion nor analysis of the power of the benthic community study. This should be done, and I suspect it will find that the power of this study is low. That is not necessarily the fault of those conducting the survey; it is an all too common problem with benthic community data in general, particularly for sites with heterogeneous substrate and contamination. This needs to be addressed in the discussion of the data and of uncertainties. If the benthic community study has low power, then it is prone to underestimating effects and is in fact a weak line of evidence rather than a strong one.

47. On page 6-22 there is discussion of selection as a mitigating circumstance for the benthic community. That is a two-edged sword, and not necessarily a mitigation against risk. Many studies have shown loss of genetic diversity associated with selection for contaminant resistance and, while it is difficult to prove, this observation can easily be extended to argue that selection for contaminant resistance decreases a population's ability to withstand other stressors. It would be best to just call this issue

a draw, inconclusive either way.

Specific Comments

1. **Figure 2-3:** The area of impacted sediments should be presented using total PAHs not just naphthalene.

2. **Tables 3-1 and 3-2 and Tables 5-1, 5-2, 5-3:** When presenting organic carbon normalized data, the organic carbon content should also be provided.

3. **Section 3.6:** Screening of constituents of potential concern (COPCs) based on the 95th percentile upper confidence limit on the mean concentration (95UCL) should not be done. All chemicals whose maximum detected concentration exceeds the screening level should be carried through the BERA risk characterization. The 95UCL should only be used in the risk characterization. In addition, after screening with the maximum concentrations, chemicals that are bioaccumulative should be retained in the BERA (e.g., mercury) even if present below screening levels. Based on the RI/FS Work Plan (Section 4.3.6.2.1.2.1), COPCs from the earlier risk assessments that would be retained as COPCs were benzo(a)pyrene, benzo(a)anthracene, xylenes, ethylbenzene, cyanide, copper, lead, mercury, and zinc. The COPCs evaluated in the BERA should begin with this list and add new COPCs based on the screening.

4. **Section 3.8.2:** Provide the total acreage of upland habitat.

5. **Table 3-9 and Section 3.11.2.2, Page 3-23 and 3-26:** It is not clear why Table 3-9 states that exposure to chemicals by fish via food transfer will not be evaluated quantitatively, but section 3.11.2.2, which discusses measurement endpoints related to Assessment Endpoint #2, includes a comparison of tissue levels of PAHs and estimated VOCs in fish to the No Effects Body Residue (NEBR). Please clarify.

6. **Section 3.11.2.3, Page 3-27, second bullet:** In order to reduce the amount of uncertainty in the black duck food chain model, it is recommended that plants are included in the dietary composition of the black duck.

7. **Section 3.11.3:** The receptors of concern (ROCs) for the aquatic habitat include a bat and tree swallow as insectivorous receptors. These species do not ingest sediment. Aquatic-dependent species that ingest sediment while foraging/nesting should also be evaluated as a ROC.

8. **Section 5 and Appendix I:** The site-specific BSAFs should be based on the 95UCL concentration not on the geometric mean.

9. **Section 5.1.2, Page 5-8:** This section discusses the possibility that fish might metabolize PAHs to more toxic metabolites. The first sentence in the first full paragraph states that detoxification is the major fate for PAHs should be revised to make it clear that while detoxification MAY occur, it is not the only possible outcome of PAH metabolism in fish.

10. **Section 5.1.2.2, Page 5-16:** The statement that claims that numerous studies show that the Critical Body Residue (CBR) provides a better estimate of toxic concentrations than sediment or surface water benchmarks should have references. In addition, this statement may not be correct because fish metabolize PAHs rapidly and thus sediment and surface water concentrations are useful measures of exposure.

11. **Section 5.1.3, Page 5-24, last paragraph:** The statement that resins and asphaltenes are non-toxic is incorrect. The organic compounds that are found in these substances can be released and can be toxic.

12. **Section 5.1.3.1, Page 5-25, first paragraph:** The document states that worms will be used as surrogates for invertivorous (invertebrate eating) wildlife. This does not make sense from an ecological or modeling standpoint as worms are in different trophic levels from birds or mammals that consume invertebrates. The statement should be corrected.

13. **Section 5.1.3.2, Page 5-26, Mammals:** The use of naphthalene to represent all PAHs is not wholly acceptable. This compound may not be the most toxic; therefore an analysis using naphthalene to represent all PAHs may not be the most conservative technique for analyzing effects of PAHs on mammals. Secondly, there are additive effects of PAHs in mixtures which may not be reflected in this technique.

14. **Section 5.1.3 and 5.1.4:** The proposed TRVs need to be re-evaluated. Use EPA Region 9 BTAG low and high TRVs as the primary source of TRVs. Secondary sources can be consulted if the COPC is not listed by Region 9 BTAG. When using the Ecological Soil Screening Level toxicity data, use both the NOAEL and LOAEL; do no extrapolate a LOAEL using a conversion factor of 5. This also applies to the other studies; a LOAEL from the literature should be selected and used, preferably from the same study if available, rather than a conversion factor of 5. A conversion factor is appropriate when a LOAEL is not available in the literature.

15. **Section 5.1.4.2, pg. 5-28:** The document states that the use of BSAF (biota-to-sediment-accumulation-factors) is "an unreliable way to evaluate the potential for adverse effects." While there is a degree of uncertainty inherent in modeling exposure, this does not necessarily make the technique unreliable. Second, the statement that "Most studies have shown that the major exposure pathway for fish to metals..." does not have references, and is therefore unsubstantiated. References should be included. Third, the last sentence in the section states that "Since there were no exceedences of screening benchmarks for metals in surface water, there is little reason to believe that metals would be elevated significantly above normal levels in Site fish." This statement is unsubstantiated (at least at this point in the risk assessment). Some metals, particularly mercury, bioaccumulate, which is why food web exposure models are done. Bioaccumulation potential needs to be discussed here.

16. **Section 5.2.2:** Surface water intake should be quantified for all higher level receptors evaluated through food chain modeling.

17. **Section 5.6 and Appendix I:** What data set was used to develop soil and sediment EPCs? Sediments far from shore should not be included in EPCs used for quantifying sediment ingestion by higher level ecological receptors, as these organisms are not expected to be exposed to sediments far from shore. Was the SEH data included in the sediment EPC dataset? Data from what soil and sediment depths was used? Why do the BERA and HHRA soil data sets for Kreher Park differ?

18. **Table 5-12:** The estimated tissue concentrations are presented as wet weight in this table, and dry weight in the Appendix I table. Ensure that dry weight tissue concentrations are used in the intake calculations as the ingestion rates used are on a dry weight basis.

19. **Table 6-1:** Individual sample locations (not an average concentration) should be compared to TECs/ PECs.

20. **Section 6.2.1, Page 6-2 and Appendix B:** PEC-quotients should be developed for each individual sediment sample to evaluate cumulative impacts from metals and PAHs.

21. **Section 6.2.1.6, Page 6-5, second full paragraph:** Is there evidence to substantiate the claim that the levels of site chemicals are higher in the top two or three inches of sediments than in the top six inches? If there are data supporting this claim, they should be referenced and discussed. Otherwise this statement is conjecture. This comment also applies to Appendix B, Section 4, Page 4-3, first paragraph.

22. **Section 6.2.13, Page 6-13, last paragraph:** This paragraph discusses evolutionary adaptations and relative susceptibilities to chemical exposure by organisms at different trophic levels. It is not necessarily true that "lower" aquatic organisms would not be adversely affected by chemical exposure if it were shown that "higher" organisms were not significantly affected by the same levels of chemical contaminants. In some cases, some "lower" groups are more susceptible than "higher" groups. This paragraph should reflect this possibility.

23. **Table 6-3, Page 6-18:** The statement that LOAELs are more reliable than NOAELs is not necessarily correct. The use of LOAELs versus NOAELs depends on the situation. The reliability of NOAELs is not in question. The statement should be reworded.

24. **Section 6.3.3.3, Page 6-25:** The three statements are misleading. Bioconcentration factors and biota sediment accumulation factors, while limited, are available in the literature for inorganics, and literature-based BSAFs were used in the document (Appendix F). Copper and selenium are known to occur at MGP sites, and both are known to be toxic to mammals and birds at concentrations slightly above nutritional requirements. While uptake into fish and invertebrate tissue from sediments or surface water may not be able to be quantified for COPCs (an uncertainty that potentially will underestimate risk), risks to aquatic receptors who forage along the shore and incidentally consume sediments contaminated with metals needs to be evaluated in this BERA.

25. **Appendix B:** Attachment 2. For the bioassay results, also present the following information in the summary tables:

Sediment Quality Metrics
Chemical/Physical
TOC (%)
AVS (umol/g)
% Fines (<63 um)
Mercury (ppm)

Empirical
PEC-Q metals
PEC-Q PAH
Mean PEC-Q
LRM Pmax
Mechanistic
ESBTU
(SEM-AVS)/foc

Toxicity Metrics
Hyalella azteca
Survival
Growth
Temperature °C (Day 0)
pH (Day 0)
Ammonia (total mg/l NH_3 - Day 0)
Ammonia (unionized mg/l NH_3)

Chironomus dilutus
Survival
Growth
Temperature °C (Day 0)
pH (Day 0)
Ammonia (total mg/l NH_3 - Day 0)
Ammonia (unionized mg/l NH_3)
Pimephales promelas
Survival
Growth
Temperature °C (Day 0)
pH (Day 0)
Ammonia (total mg/l NH_3 - Day 0)
Ammonia (unionized mg/l NH_3)

26. **Appendix B, Section 4, Page 4-2, 1st paragraph, last sentence:** This statement is misleading and should be rewritten. The LSRI report noted that the survival in the performance control samples for *C. dilutus* and the minnow met acceptability criterion. The minnow showed significant effects on growth at SQT1 and under UV light there was significant reduction in survival at SQT1 and SQT7. Overall, it appears that the reference stations have been impacted and comparisons to performance control samples should be given more weight than comparison to the reference samples.

27. **Appendix B, Table 3-1:** Why was the organic carbon content of SQT3 (sand) so high (40%)? This percentage is similar to the highest concentrations found in the wood stations (42%). Should this location be excluded from the sand samples and included as a wood station?

28. **Appendix B, Attachment 2:** NOEC and LOEC values presented in the tables and text do not match. Values in Tables 1-6, 3-1, and 5-2 need to be verified with the text. For instance, text on page 3-1 does not match numbers in Table 3.1. "Average NOECs ranged from 735 to 8031. Should 8031 be 7257 µg PAH/gOC as presented in Table 3.1? Also change 139.5 µg/g to 135.1. In last sentence, should 3,396 be changed to 3,996.

29. **Appendix B, Attachment 2, Section 1.4, Page 1-6:** The statement that no NOECs or LOECs are proposed for *H. azteca* contradicts the tables and discussion in the main text (Section 5.1.2.2) where NOECs and LOECs are presented, discussed, and (presumably) incorporated into the average and range of NOECs and LOECs. In addition, the statement on 3-1 (Appendix B, Attachment 2, Section 3) that describes the range of average NOECs (735 to 8031 ug PAH/gOC) also is in disagreement with the statement in section 5.1.2.2, pg 5-18 that describes the range of average NOECs (735 to 7257 ug PAH/gOC). The values should be corrected to be consistent.

30. **Appendix C and Appendix I:** The fish tissue concentration in Appendix C is on a wet weight basis; the 95UCL tissue concentration presented in Table I-3 is on a dry weight basis. Please clarify which is correct. A summary table of fish tissue data should be provided in the text similar to the summary tables for sediment and soil presented in Appendix A. How was the 95% UCL concentration of total PAHs in fish calculated?

31. **Table F-1:** The 90th percentile percent soil in the diet of a dove of 13.9% (EPA, 2005) should be applied for the blackbird.

32. **Table F-2:** The regression model in EPA 2005 should be used to estimate soil invertebrate concentrations for cadmium. Provide soil BAFs for dibenzo(a,h)anthracene. EPA 2005 provides a regression model for total PAH uptake into foliage. The BSAFs for sediment invertebrates and fish should be based on 95UCL tissue concentrations not the geometric mean.

33. **Appendix I:** Dose tables should provide all intake factors used for the receptor including the BSAFs, prey intake rates, soil intake rates, and body weight. For the osprey, cormorant, and mink present the measured fish tissue concentrations.

Typos and Editorial Errors

34. **Section 2.3, Page 2-2, first sentence:** Sentence should read: "The Site is located at the top of a ravine on the shore of Chequamegon Bay."

35. **Section 3.2.2, Page 3-2, second paragraph:** The statement: "SEH (2002) reported that the levels..." is not relevant to an Ecological Risk Assessment. Delete this statement.

36. **Section 3.8.1.2, Page 3-13, last paragraph:** Sentence should read: "...the list of those species frequenting the Site waters..."

37. **Section 5.1.2.2, Page 5-17, second full paragraph:**
a) Sentence should read: "The results..."
b) Section on Hyallela; Sentence should read: "Furthermore, the mortality was consistent..." pg. 5-18, Section on Chironomus, last paragraph; and Tables 5-1 to 5-3
c) This paragraph needs clarification and rewriting. The statement beginning "When the NOECs and LOECs..." needs proper punctuation and clarification. Are the NOECs/LOECs for each species averaged separately or lumped together? The statement should read that the NOECs for each species are averaged between the 2001 and 2005/6 results, correct?
d) The average LOEC for H. azteca is 422.8 ug/g, not 453.3 ug/g. Therefore, the range of average LOECs is 79.9 ug/g to 422.8 ug/g, not 208.3 ug/g. The average LOEC for total PAHs per gOC for H. azteca is 5463 ug/OCg, not 11494 ug/gOC.
e) pg. 5-20, first full paragraph: Sentence should read: "The unionid snail, also an epibenthic species, was absent from all of the Site..."

38. **Section 5.1.4.3, Page 5-30, Selenium:** The sentence: "However, both laboratory and field studies..." should read "accumulate egg selenium concentrations much greater than 3 mg Se/kg dry weight without adverse effects on reproduction." (delete "are").

39. **Section 6.2.1.2, Page 6-3:** The sentence "At the other stations..." is not clear. Please rewrite for clarity. Also, use of contractions is not appropriate. (See also Section 6.2.1.5, pg. 6-4).

40. **Section 6.2.1.4, Page 6-3:** The first sentence should read "...indicated that there were significant effects..."

41. **Section 6.2.2, Page 6-6, first numbered bullet:** Sentence should read: "Compare ~~of~~ concentrations of Site-related..." (delete "of").

42. **Section 6.2.14, Page 6-14, second paragraph:** Second to last sentence should read "thick, dull iridescence with brown streaks about..."

43. **Section 6.3.2, Page 6-18, last paragraph:** Sentence should read: "The information presented, while as complete and accurate as possible, may have missed...". It is extremely unlikely that in any scientific study, information will be complete, especially in a Superfund site.

If you have any questions or would like to discuss things further, please contact me at (312) 886-1999.

Sincerely,

Scott K. Hansen
Remedial Project Manager

cc: Dave Trainor, Newfields
Jamie Dunn, WDNR
Dave Mount, EPA
Omprakash Patel, Weston Solutions, Inc.
Ervin Soulier, Bad River Band of the Lake Superior Chippewa
Melonee Montano, Red Cliff Band of the Lake Superior Chippewa

TABLES AND FIGURES

Table 1. BSAFs based on total PAH for sand and wood sites reported by URS (2006).

Site	Type	TOC (%)	PAH (ug/g OC)	Geo Mean Tissue PAH (ug/g lipid)	BSAF (lipid/OC)
SQT6	Sand	9.5	24	71.5	2.98
SQT3	Sand	40	43	430	10.00
SQT2	Wood	42	7.6	32.2	4.24
SQT4	Wood	42	34	189	5.56
SQT5	Wood	25	125	597	4.78
SQT8	Wood	28	250	37.8	0.15

Table 2. 10-day LC50 and LC20 concentrations calculated from exposures of *Hyalella azteca* to dilutions of SQT1 (URS 2006), with and without UV and shading (leaf plugs).

Treatment	LC50 (ug PAH/g OC)	LC20 (ug PAH/g OC)
Lab Light	12748	9593
UV Light (unshaded)	6052	4557
UV Light with leaf plugs	9544	7676
UV Light corrected for non UV toxicity (Lab Light)	6143	4562
UV Light with leave plugs corrected for non UV toxicity (Lab Light)	10164	8359

Table 3. PAH concentrations predicted to cause 20% mortality in 10 days as a function of UV exposure at depth, based on URS (2006) laboratory exposures.

Depth (cm)	PAH conc Predicted to Cause 20% Mortality in 10 days at	
	No Shade	Shaded (leaf plugs)
25	216	396
50	312	572
75	451	827
100	652	1195
125	943	1727
150	1363	2497
175	1969	3609
200	2847	5216
225	4115	7540
232	4562	8359
250	5948	10898
275	8597	15753
300	12427	22770